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Double-Chambered Left Ventricle Complicated with Thrombosis Presenting with Unilateral Limb Weakness as the Initial Manifestation: A Two-Case Report

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Abstract

Double-chambered left ventricle (DCLV) is a rare congenital heart disease. Its clinical manifestations in children are often subtle and may be easily overlooked or misdiagnosed. This case report describes two children with type A DCLV complicated by ventricular thrombosis and cerebral infarction who presented with unilateral limb weakness as the initial symptom. Case 1 involved a 5-year-old boy who developed right-sided limb weakness after convulsions and was subsequently diagnosed with cerebral infarction. The primary disease was DCLV complicated with thrombosis. During the waiting period for the surgery, the right hemiplegia worsened. The emergency cranial CT indicated that the cerebral infarction had worsened with a small amount of bleeding. After the acute phase of intracranial hemorrhage had resolved, surgical correction of the DCLV and thrombus removal were performed. The patient recovered well after the operation. Limb movement returned to normal during nearly 3 years of follow-up. Case 2 involved a 1-year-6-month-old boy who suddenly experienced right-sided limb dysfunction. After examination, it was found that it was a case of DCLV combined with thrombus detachment into the cerebral blood vessels.

Emergency cardiac surgery was performed, and limb function partially improved 8 months after the operation. For children presenting with unexplained unilateral limb paralysis and acute cerebral infarction, cardiac screening should not be overlooked. The timing of surgery should be individualized according to the intracranial condition, and combined treatment involving malformation correction, thrombus removal, and postoperative rehabilitation should be implemented to improve long-term outcomes. This study reports only 2 cases, and further large-sample studies are needed to explore the clinical characteristics and long-term prognosis of this disease.

1.Introduction

Double-chambered left ventricle (DCLV) is an extremely rare congenital heart disease with an unclear definitive incidence, and its prevalence rate ranges from 0.04% to 0.42% [1,2]. Patients with DCLV present with nonspecific clinical manifestations. Patients may be asymptomatic or exhibit atypical symptoms such as palpitations, chest tightness, and shortness of breath after any activity, and some severe cases may progress to left-sided heart failure and pulmonary edema. The clinical manifestations of pediatric DCLV cases are particularly atypical, and most cases are detected incidentally during physical examination. However, concurrent heart failure may be life-threatening [3]. This case report describes two children with DCLV who presented with unilateral limb weakness as the initial symptom and were admitted to our cardiac center. The clinical characteristics and treatment regimens of the disease are discussed in conjunction with a literature review. The case report provides a reference for the clinical diagnosis and treatment of DCLV.

2. Case Presentation

Case 1

A 5-year-old male child was admitted to the hospital because of convulsions accompanied by right-sided limb weakness for 2 days. The child developed unprovoked convulsions that lasted for approximately 5 minutes and resolved spontaneously, followed by persistent right-sided limb weakness. Emergency cranial magnetic

resonance (CMR) imaging performed at an external hospital revealed the following: 1. dissection with an intramural hematoma in the superior quadrant of the M1 segment of the left middle cerebral artery, involving the ostia of the partial lenticulostriate arteries, with moderate-to-severe stenosis of the corresponding lumen; and secondary acute cerebral infarction with slight hemorrhage in the left basal ganglia and corona radiata. 2. focal cerebral atrophy and abnormal signals in the right frontoparietal lobe, suggestive of sequelae of encephalitis. The child was admitted to the Department of Neurosurgery at our hospital for further evaluation and treatment. Subsequent echocardiography confirmed type A DCLV, thrombotic echoes in the left and right ventricles, left ventricular noncompaction, and a small amount of pericardial effusion (Figure 1A). The patient was immediately transferred to the Department of Cardiac Surgery. He had been previously healthy, with no remarkable personal or family history.

Physical examination at admission revealed the following: temperature of 36.3 °C, heart rate of 108 beats/minute, respiratory rate of 21 breaths/minute, blood pressure of 93/67 mmHg, and body weight of 23 kg. The child was conscious, alert, and able to answer questions appropriately. Bilateral pupils were equal and round with normal light reflexes, and frontal wrinkles were symmetrical. The left palpebral fissure was narrower than the right palpebral fissure, and the left nasolabial fold was shallower than the right fold. The tongue protruded in the midline. Increased muscle tone was noted in the right lower limb, and muscle strength in the right upper limb was grade I. Muscle strength and tone in the left limbs were normal. The child could sit and stand independently for a brief period.

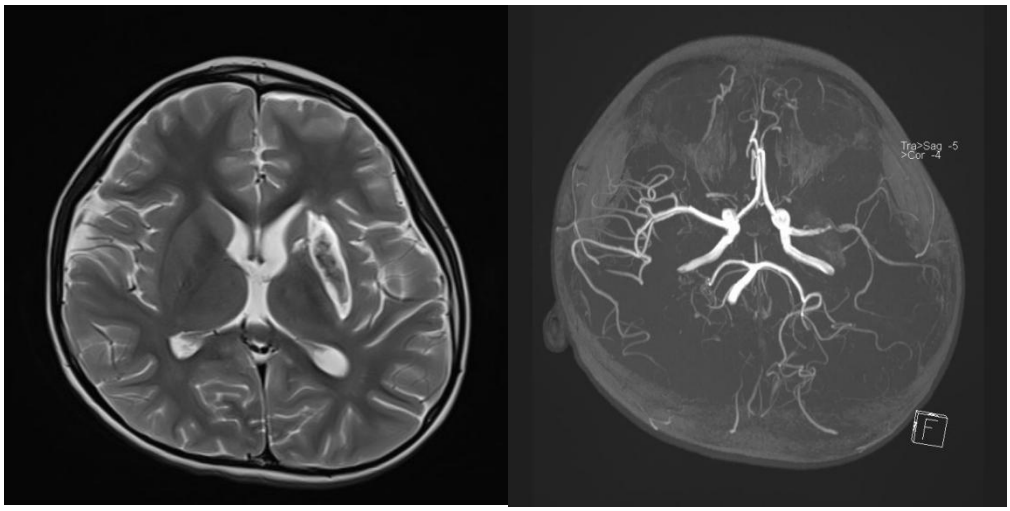
Auxiliary examinations revealed that routine blood tests, rapid C-reactive protein levels, coagulation function, D-dimer levels, and cardiac, hepatic, and renal function were all within normal ranges. Cardiac computed tomography (CT) revealed findings suggestive of DCLV(Figure 1B) . CMR imaging demonstrated hemorrhagic cerebral infarction in the left basal ganglia and left cerebral peduncle, moderate-to-severe stenosis of the M1 segment of the left middle cerebral artery with significantly sparse distal branches, and occlusion of the P3 segment and distal branches of the right posterior cerebral artery (Figure 1C, D). The final diagnoses were type A DCLV,

ventricular thrombosis, intracranial hemorrhage, cerebral infarction secondary to cerebral arterial thrombosis, and hemiplegia.

On the second day after transfer to the cardiac surgery department, the child developed vomiting, lethargy, and aggravated right-sided limb weakness. Emergency cranial CT indicated cerebral hemorrhage in the left internal capsule and basal ganglia. An emergency neurosurgical consultation was obtained, and craniotomy was not indicated because of the limited volume of intracranial hemorrhage. Neurological management consisted of neurotrophic supportive treatment. Cardiac surgery was temporarily postponed because of the acute phase of intracranial hemorrhage and contraindications to extracorporeal circulation. Approximately one week later, surgical correction of the DCLV combined with thrombus clearance was successfully performed. After recovering from cardiac surgery, the child received hyperbaric oxygen therapy and rehabilitation at an external medical institution. At nearly 3 years and 1 month postoperative follow-up, right-sided limb function had fully recovered, and the child could perform normal activities of daily living.



A B



C D

Figure 1 Case 1 patient. A: An echocardiogram shows that the two left ventricular chambers are separated by an abnormal muscle bundle. B: Cardiac computed tomography revealed findings suggestive of DCLV. C: A CMR image shows hemorrhagic cerebral infarction in the left basal ganglia and left cerebral peduncle. D: Moderate-to-severe stenosis of the M1 segment of the left middle cerebral artery with significantly sparse distal branches, and occlusion of the P3 segment and distal branches of the right posterior cerebral artery

Case 2

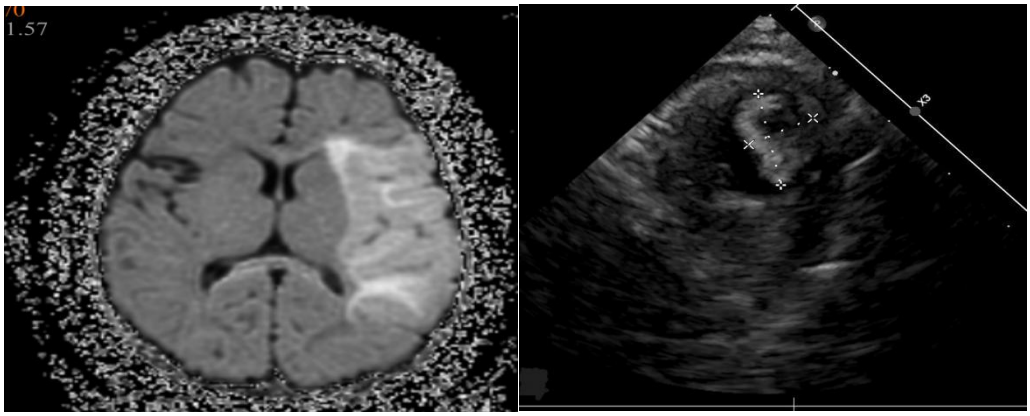
An 18-month-old male child was admitted with a 3-day history of right-sided limb dysfunction. Three days prior to admission, the patient developed right upper limb weakness, an unsteady gait, and paroxysmal twitching of the angle of the mouth following crying. Emergency cranial CT performed at a local hospital revealed large hypodense lesions in the left frontotemporoparietal lobe and localized increased density of the left middle cerebral artery, which were highly suggestive of the left middle cerebral artery territory with extensive cerebral infarction. CT imaging confirmed abnormal signals in the left temporoparietal region consistent with acute cerebral infarction and occlusion of the left middle cerebral artery (Figure 2A). Echocardiography demonstrated a left ventricular apical diverticulum with a mobile thrombus and a small amount of pericardial effusion. The child was transferred to our hospital for further evaluation and treatment. The patient had no remarkable personal or family medical history.

Physical examination at admission revealed the following: temperature of 37.4 °C, heart rate of 173 beats/minute, respiratory rate of 24 beats/minute, blood pressure of

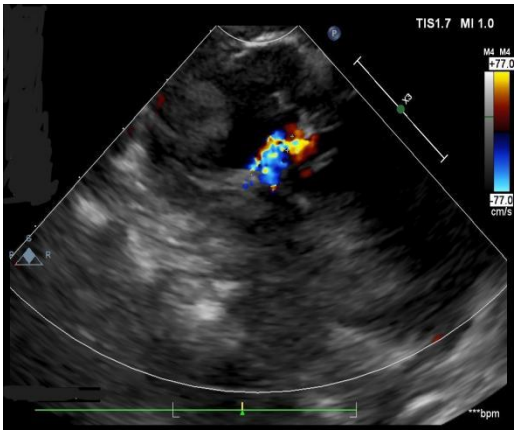
88/49 mmHg, and body weight of 11 kg. The child was conscious, alert, and able to follow instructions and communicate normally. Bilateral pupils were equal and round with brisk pupillary light reflexes. No abnormal pulmonary or abdominal findings were noted. The heart sounds were strong and regular, with no precordial murmur. Physiological reflexes were present, and pathological reflexes were not elicited. Muscle strength and tone in the left limbs were normal. The right upper limb had grade III muscle strength and decreased tone, and the right lower limb had grade III and IV muscle strength and slightly reduced tone. Hyperactive right-sided tendon reflexes and a suspiciously positive Babinski sign were observed.

Auxiliary examinations revealed that routine blood tests; rapid C-reactive protein levels; coagulation function; D-dimer levels; and cardiac, hepatic, and renal function were all within normal limits. Echocardiography revealed multiple abnormal muscle bundles in the anteromedial and inferior segments of the left ventricle, which led to the compartmentalization of the mid- and inferior left ventricular cavity. Multiple communications were identified between the basal abnormal cavity and the native left ventricular cavity, with the largest orifice measuring approximately 2.2 mm in width. A thrombus-like echogenic mass measuring 12.5 × 8.4 mm was detected at the left ventricular apex, which was characterized by peripheral hyperechoic signals and internal anechoic areas (Figure 2B, C). The final diagnoses were type A DCLV, ventricular thrombosis, cerebral infarction, and hemiplegia.

Symptomatic supportive treatments, including dehydration and neurotrophic therapy, were administered. Combined emergency consultation involving neurology and neurosurgery recommended no invasive neurological intervention and emphasized the need for early cardiac surgical treatment. Emergency surgical correction of the DCLV combined with thrombus removal was performed on the day of admission. At the 8-month postoperative follow-up, the motor function of the patient’s right limb had significantly improved.



A B



C

Figure 2 Case 2 patient. A: A CT image confirms abnormal signals in the left temporoparietal region consistent with acute cerebral infarction. B: A thrombus-like echo can be observed at the apex of the left ventricle. C: Orifice of the intracavitary communication within the left ventricle

3. Discussion

The pathogenesis of DCLV remains unclear. Some scholars believe that it results from excessive hyperplasia or incomplete regression of the trabeculae in the mid-ventricular or apical region during the embryonic period [4]. In a case series of 12 patients with DCLV, Zhang et al. [5] found that 5 of the patients were relatives and that all the subjects harbored MYH7 mutations, which are known to be associated with hypertrophic and dilated cardiomyopathy.

DCLV is a congenital cardiac anomaly in which abnormally hypertrophied muscle bundles or fibrous bands divide the left ventricle into two cavities, namely primary and accessory chambers. Typically, the primary chamber is located in the proximal basal segment that encompasses mitral and aortic orifices, and the accessory chamber is

located in the apical or lateral wall of the distal ventricle. According to the spatial arrangement of the two cavities, DCLV can be classified into two types: type A (superior–inferior arrangement) and type B (left–right arrangement). Most of the reported DCLV cases are of type A [6], which is consistent with the diagnoses of the two cases in the present study. DCLV can occur as an isolated malformation or be combined with other congenital cardiac defects, such as atrial septal defects and tetralogy of Fallot [2].

The therapeutic strategy for DCLV depends on left ventricular function and the presence of concomitant cardiac malformations. DCLV without significant hemodynamic abnormalities can be managed with regular clinical follow-up [6]. Meng et al. [4] reported seven cases of type A DCLV without severe ventricular outflow obstruction or left heart failure. For all these cases, favorable long-term outcomes were achieved with conservative management. In contrast, the two pediatric patients in our study developed intracavitary ventricular thrombosis. Thrombus detachment can lead to acute cerebral infarction and severe extracardiac complications, necessitating early surgical intervention. Surgical correction eliminates the structural abnormality and clears the intracardiac thrombus at the source, effectively preventing recurrent thromboembolism and new-onset cerebral infarction. The formation of a intracavitary thrombus may be attributed to blood flow stasis and reduced flow velocity across the stenotic orifice between the primary and accessory chambers, which ultimately promotes thrombus deposition within the ventricular cavity [3,7].

Pediatric acute ischemic stroke (AIS) is a rare but severe neurological emergency, with an incidence of 0.1%–0.2%. According to the 2019 American Heart Association/American Stroke Association (AHA/ASA) guidelines for pediatric stroke management [8], congenital and acquired heart diseases as well as arrhythmias are categorized as the major cardiogenic causes of pediatric AIS. Cardiogenic factors, including congenital heart defects, surgery-related complications, or acquired cardiac diseases, account for approximately 30% of all pediatric AIS cases. Notably, pediatric AIS differs significantly from adult AIS in terms of clinical manifestations, risk factors, pathophysiological characteristics, reperfusion responses, and preventive and

therapeutic strategies [8,9]. Therefore, the efficacy and safety of intravenous thrombolysis and mechanical thrombectomy in children remain controversial. Neither of the two patients in our study received cerebrovascular thrombolysis before or after cardiac surgery. Both patients were treated with conservative medical management and standardized postoperative rehabilitation, which resulted in a satisfactory overall prognosis.

Extremely high rates of misdiagnosis and missed diagnosis have been reported for pediatric DCLV with initial neurological thromboembolic manifestations, primarily because of limited clinical awareness and insufficient standardized screening protocols. In this study, the two patients did not exhibit typical cardiac murmurs, heart failure, or cardiac enlargement. When treating children with hemiplegia and acute cerebral infarction, clinicians tend to focus solely on neurological etiologies, neglecting occult congenital cardiac structural abnormalities. The common differential diagnoses for pediatric acute cerebral infarction include intracranial and extracranial arterial lesions, thrombophilia, sickle cell disease, and autoimmune diseases [8]. On the basis of the two cases described here, we emphasize that routine cardiac screening should be performed for children with unexplained acute cerebral infarction or unilateral limb hemiplegia without definite trauma, infection, or autoimmune causes, to facilitate the early identification of occult DCLV and intracardiac thrombosis. Individualized surgical timing formulated according to the status of intracranial lesions, combined with radical correction of the cardiac malformation, thorough thrombus removal, and standardized postoperative rehabilitation, may effectively reduce neurological sequelae and improve long-term outcomes in pediatric patients.

4. **Limitations**

This case report has a small sample size of only two patients. Additional large-sample and long-term follow-up clinical studies should be conducted to systematically summarize the clinical characteristics, risk factors, and prognostic factors of pediatric patients with DCLV complicated with thromboembolism. In addition, the follow-up duration differed between the two children, and their long-term cardiac function and neurodevelopmental outcomes require continued prospective observation

5. Conclusion

DCLV is a rare congenital cardiac malformation. Pediatric patients usually present with insidious and atypical cardiac manifestations, leading to a high risk of misdiagnosis. In this study, both children initially presented with neurological deficits characterized by unilateral limb weakness. Structural abnormalities of the left ventricle resulted in intracardiac blood stasis and subsequent thrombus formation and detachment, leading to severe cardiogenic stroke. Timely surgical correction and thrombus removal, combined with standardized postoperative rehabilitation, can significantly improve neurological function and long-term outcomes. Clinicians should maintain a high index of suspicion for this rare disease and perform routine cardiac screening in children with unexplained cerebral infarction to facilitate the early detection of occult DCLV.

Declarations

Abbreviations

DCLV	Double-chambered left ventricle
CMR	Cranial magnetic resonance
CT	Computed tomography
AIS	Acute ischemic stroke
AHA/ASA	American Heart Association/American Stroke Association

Ethics approval and consent to participate

This study was performed with the ethics approval from the Institutional Committee of Guangzhou Women and Children's Medical Center, Guangzhou Medcial University (Approval NO. 212A01.). The study obtained informed consent for publication of clinical details and clinical images was obtained from the parents of the patient.

Availability of data and materials

No data and materials were generated or analysed during the current study.

Competing interests

The authors declare no competing interests.

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Author Contributions

Jia Yuan wrote the manuscript text, Mingjie Zhang and Bingjie Fan prepared figures, Li Ma was responsible for the surgery, Na Zhou designed the manuscript. All authors reviewed the manuscript and criticaally edited it.

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Clinical trial number

Not applicable

Informed Consent

As our case report does not meet the definition of research, it is not subject to institutional review board (IRB) oversight. As such informed consent was not obtained from the individual participant included in the case report.

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